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Clinical Neuropsychology

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Jim Gilligan, Esquire
US Department of Justice
US Attorney, Western District of Texas

Exhibit A

Re: Jerome Schmidt, Ph.D.

Dear Mr. Gilligan,

By way of qualifications, I am currently Clinical Professor in the Department of Psychiatry and Biobehavioral Sciences at UCLA, and have been since 2008. I have held a Diplomate in Clinical Neuropsychology since 1995, and am a Fellow of the American Psychological Association and the National Academy of Neuropsychology. I am licensed as a psychologist in California. I have authored and co-authored extensively in neuropsychology as evidenced in my curriculum vita. I have reviewed the medical records, expert reports, and depositions regarding Jerome Schmidt, M.D., and the following are my comments and opinions:

Austin Center for Therapy and Assessment:

The plaintiff underwent neuropsychological evaluation on November 16, and 25, 2015, under the supervision of Wayne Dees, Psy.D.; testing was apparently conducted by a masters level associate (Claire Allen, M.A.). At that time the plaintiff was reporting severe problems in memory since an October 13, 2015, motor vehicle accident, as well as moderate problems in reading comprehension, word finding, and remembering names, and mild problems in oral comprehension and concentration. He was additionally describing moderate depressive symptoms, and mild to moderate anxiety. Neuropsychological test results were interpreted as showing evidence of overall low average verbal and visual memory (with some scores in the borderline impaired range) and decreased complex tactile discrimination, while all other scores were average or higher; screening of overall intelligence revealed a superior range score (120; 91st percentile). On self-report psychiatric symptom inventories, mild depression and significant anxiety were found. It was concluded that the evaluation results were

"consistent with symptoms of Major Neurocognitive Disorder, secondary to TBI. There is evidence of significant cognitive decline from a presumed previous level of

cognitive functioning. His cognitive deficits interfere with daily functioning, including work activities and concentration, and are consistent with fronto-temporal lobe impairment. In addition, Dr. Schmidt appears to meet diagnostic criteria for an Adjustment disorder with depression, and a Generalized Anxiety disorder. Although previously diagnosed with PTSD, he does not appear to be exhibiting any significant symptoms of this disorder presently."

However, there are concerns regarding the conclusions from that exam, as follows:

1) No performance validity tests (PVTs) were administered to verify that the plaintiff was performing to true ability on testing. Use of PVTs is mandated in a neuropsychological exam, particularly in a forensic context (see American Academy of Clinical Neuropsychology Consensus Conference Statement on the Neuropsychological Assessment of Effort, Response Bias, and Malingering, 2009), but also in clinical contexts, and certainly when test takers stand to gain from symptoms (e.g., financial compensation). Recent survey data have shown that among practicing neuropsychologists, the average number of PVTs used in a forensic exam is six, and the average number employed in a clinical exam is five (Martin et al., 2015); thus, Dr. Dees' exam, which included no PVTs, falls well below current practice in the field. Without data from PVTs to document that the plaintiff was in fact performing to true ability, test scores can not be used as evidence of cognitive abnormality.

2) No appropriate personality testing was conducted. Psychiatric symptom self-rating scales were employed (Beck Depression Inventory – II; State Trait Anxiety Inventory), but these do not contain validity scales to verify that the test taker is providing reliable data regarding symptoms. A more appropriate choice, and the personality inventory that is used most often by neuropsychologists to verify validity of symptom report (see Martin et al., 2015), is the Minnesota Multiphasic Personality Inventory – 2 – Restructured Form (MMPI-2-RF). This instrument, published in 2008, contains several over-report validity scales to check as to whether the test taker is over-reporting psychiatric, but also physical and cognitive, symptoms in a nonplausible manner. Additionally, the test contains multiple clinical scales to assess as to whether a test taker has psychiatric conditions over and above depression or anxiety. In the plaintiff's case, there is some evidence that he engages in hyperbole regarding symptoms (e.g., he described his memory problems as "severe" when in fact overall memory performance was low average). Such "catastrophizing" in regards to symptom report is found in a somatoform disorder; in a somatoform disorder the individual channels stress into physical symptoms, magnifies the seriousness of minor symptoms, and views him- or herself as more dysfunctional than is actually the case. The MMPI-2-RF would have provided data relevant to this issue, and without the MMPI-2-RF or similar measures, Dr. Dees does not know, and has not ruled out, a somatoform disorder as leading to symptom overreport. A somatoform disorder could account for the long term postconcussive symptoms described by the plaintiff. The 2015 accident would not have "caused" a somatoform disorder; as documented by Greiffenstein and Baker (2001), elevations on hypochondriasis and somatic symptom scales are found to *pre-date* concussive

injury in individuals claiming longterm residuals from concussion, and as such reflect a longstanding, characterological disposition.

3) Dr. Dees concluded that the plaintiff has a cognitive disorder "secondary to TBI," but at the time of the motor vehicle accident the plaintiff did not lose consciousness, was coherent at the scene (Glasgow Coma Scale = 15), did not experience anterograde amnesia (loss of memory for events post-injury), and had no brain imaging abnormalities. Thus, there is no objective evidence of concussion/mild traumatic brain injury (criteria for concussion: loss of consciousness <30 minutes, Glasgow Coma Scale = 13-15, anterograde amnesia <24 hours, and no brain imaging abnormalities). Even if the plaintiff had experienced a concussion, reviews of the literature on neuropsychological function in mild traumatic brain injury (see Carroll et al., 2004, 120 studies; Dikmen et al., 2009, 33 studies), including six meta-analyses involving dozens of studies and thousands of patients in the aggregate (133 studies, n = 1463, Belanger et al., 2005; 21 studies, n = 790, Belanger & vanderploeg, 2005; 8 studies, Binder et al., 1997; 17 studies, n = 634, Frencham et al., 2005; 25 studies, n = 2828, Rohling et al., 2011; 39 studies, n = 1716, Schretlen & Shapiro, 2003), show that patients who experience mild brain trauma have returned to baseline by weeks to months post-injury. In a book summarizing the research on mild traumatic brain injury (McCrea, 2008), published under the auspices of the American Academy of Clinical Neuropsychology (the membership organization for board certified neuropsychologists), it is concluded that there is "no indication of permanent impairment on neuropsychological testing by three months postinjury" (p. 117).

4) The plaintiff had conditions which could more likely account for any cognitive inefficiency, such as depression, and reduced sleep and regular use of ambien (10mg once every 3 nights plus alprazolam .5mg every 3 nights). Depression (Boone et al., 1995), sleep deprivation (Benitez & Gunstad, 2012), and use of ambien (Stranks & Crowe, 2014) have been documented to lead to mild cognitive decrements.

5) Dr. Dees apparently attributed depression and anxiety to the injury in October of 2013, but research shows no long-term psychiatric conditions (i.e., depression, anxiety, poor coping ability, psychological distress) from concussion (see meta-analysis by Panayiotou et al., 2010).

6) Dr. Dees recommended various treatments, including cognitive rehabilitation, but in fact no treatment is recommended or indicated for concussion. For example, the American Academy of Neurology published guidelines in 2013 for "management" of concussion, and in that document it is noted that:

> *"Patients with mTBI/concussion may underestimate their preinjury symptoms, including many symptoms that are known to occur in individuals without concussion, such as headache, inattention, memory lapses, and fatigue. After injury there is a tendency to ascribe any symptoms to a suspected mTBI/concussion. …Cognitive restructuring is a form of brief*

psychological counseling that consists of education, reassurance, and reattribution of symptoms……..”

In other words, the “treatment” for concussion is to reassure, educate that individuals fully recover from concussion, and help patients “reattribute” claimed concussion symptoms to their correct causes. In fact, prolongation of symptoms is more pronounced when treaters/evaluators focus attention on symptoms and remove patients from life activities. For example, adolescents who were less compliant with recommendations for physical and cognitive rest after concussion recovered ***more*** quickly than adolescents who more closely followed the recommendations (Moor et al., 2015), and teenagers and young adults who followed rest recommendations reported “***more*** daily postconcussive symptoms…and slower symptom resolution” (Thomas et al., 2014). Other investigators have come to similar conclusions (“activity restriction itself may contribute to protracted recovery and other complications;” DiFazio et al., 2015). Similarly, Buckley et al. (2016) reported that prescribed physical and cognitive rest post-concussion was “not effective in reducing postconcussion recovery time” (see also McCrea et al., 2009).

7) Dr. Dees is not board certified in neuropsychology, which raises questions as to his actual level of competency in neuropsychological assessment, i.e., no official neuropsychological organization has vouched as to his competency in the field.

The plaintiff underwent repeat neuropsychological evaluation under the supervision of Dr. Dees on October 4, 2016; testing was apparently conducted by Amy Duckwall, Psy.D. At that time the plaintiff was no longer taking ambien and Xanax, but was prescribed hydrocodone (10/325); it was not reported how often he was taking the medication. In the interim between exams the plaintiff had undergone back surgery (August of 2016), and been diagnosed with sleep apnea. The plaintiff was reporting concern regarding the permanence of his “horrible” cognitive impairments; he was continuing to report problems in memory, concentration, word-retrieval, reading comprehension, poor judgment (e.g., driving aggressively), and mental confusion. Of particular note, he was stating that his memory was *worsening.* He had been receiving weekly speech therapy. Neuropsychological scores were interpreted as showing impairment (1st percentile scores) in word reading speed and visual memory, and low average scores in visual constructional ability, overall visual memory, verbal memory, motor dexterity in the left hand, and color naming speed. The plaintiff was diagnosed with mild neurocognitive disorder secondary to closed head injury in 2015, and adjustment disorder with mixed Anxiety and Depressed Mood. However, there are concerns regarding the conclusions from that exam, as follows:

1) As with the first exam, no performance validity tests (PVTs) were administered to verify that the plaintiff was performing to true ability on testing. Without data from PVTs to document that the plaintiff was in fact performing to true skill level, test scores cannot be used as evidence of cognitive abnormality. Of note, the plaintiff actually obtained some lower scores on the second evaluation as compared to the first exam (e.g., visual construction, rapid word reading) which would not be the course after a traumatic brain injury; after a brain injury, test scores remain static or gradually improve, and there would be no brain/behavior mechanism to account for

a drop in scores remote from injury. In fact, practice recommendations regarding the identification of noncredible cognitive performance published by the American Academy of Clinical Neuropsychology indicate that nonplausible changes in scores on sequential testing can be used to document response bias and suboptimal effort (Heilbronner et al., 2009).

2) As with the first exam, no appropriate personality testing was conducted. Psychiatric symptom self-rating scales were employed (Beck Depression Inventory – II; State Trait Anxiety Inventory), but these do not contain validity scales to verify that the test taker is providing reliable data regarding symptoms. Further, as discussed above, there is evidence that plaintiff engages in hyperbole regarding symptoms (e.g., in the 2016 exam he described his cognitive impairments as "horrible," when the vast majority of his scores were average or higher; and he stated that it "'scares the hell out of me' to think that his nondominant right hand might now be stronger that his left hand"). Catastrophizing in regards to symptom report is found in a somatoform disorder, and Dr. Dees did not administer the appropriate tests to identify whether the plaintiff has somatoform tendencies. A somatoform disorder could account for the long term postconcussive symptoms reported by the plaintiff.

3) Dr. Dees concluded that the plaintiff has a cognitive disorder "secondary to a closed head injury sustained in 2015." However, as discussed above, there is no objective evidence that the plaintiff even sustained a concussion, and even if he did, as summarized above, research shows no longterm cognitive sequelae from concussion/mild traumatic brain injury.

4) The 2016 report shows that the plaintiff had conditions at that time which could more likely account for any cognitive inefficiency, such as depression, sleep apnea, and use of hydrocodone. Depression (Boone et al., 1995), sleep apnea (see meta-analysis by Bucks, Olaithe, & Eastwood, 2012), and use of vicodin (Gruber, Silveri, & Yurgelun-Todd, 2007), have been documented to lead to mild cognitive decrements.

5) Dr. Dees again attributed depression and anxiety to the injury in October of 2013, but as discussed above, research shows no long term psychiatric conditions (i.e., depression, anxiety, poor coping ability, psychological distress) from concussion (see meta-analysis by Panayiotou et al., 2010).

6) Dr. Dees recommends various treatments, but as discussed above, no treatment is recommended or indicated for concussion.

Dr. Dees issued a letter (undated) to Attorney Henry Moore, plaintiff counsel, in which he responded to questions posed by Mr. Moore, as summarized below:

1) Dr. Dees was asked whether the symptoms he observed in the plaintiff were consistent with the history of a closed head injury sustained on October 13, 2015. Dr. Dees opined that they were due to coup/contrecoup and/or diffuse axonal injury, conclusions that are outside of his area of expertise. Such injuries would have been

detected on brain imaging, and would have been accompanied by loss of consciousness and altered mental status. Again, as summarized above, the results of several meta-analyses on outcome from concussion/mild traumatic brain injury (which there is no evidence that the plaintiff even sustained) show no long term symptoms.

2) Dr. Dees was asked whether the plaintiff's history of premorbid minimal difficulty with memory, PTSD, mild depression, and some difficulty with focus contained in the records from the plaintiff's family physician could explain the plaintiff's current symptom report. Dr. Dees opined that the cognitive and psychiatric symptoms worsened secondary to the accident in 2015 because "when interviewed by me in November 2015, he stated that he had noticed more problems….". Dr. Dees conducted no appropriate personality testing to document whether the plaintiff's self-report of symptoms was accurate, or to rule out the presence of a somatoform condition in which minor symptoms are magnified; the plaintiff's hyperbole when reporting symptoms is a red flag for a somatoform condition. Without appropriate personality testing to verify accuracy of symptom report, the plaintiff's report of symptoms cannot be relied upon. Dr. Dees also bases his conclusion that the plaintiff's cognitive symptoms are worse post-accident on the results of the neuropsychological testing. But as discussed above, Dr. Dees did not administer PVTs necessary to verify that the plaintiff was performing to true ability; without those data, the neuropsychological results cannot be used to document cognitive dysfunction.

3) Dr. Dees was asked whether it was significant that EMS at the scene of the October 2015 accident were told that there were no injury and that EMS were not needed, that the plaintiff did not attempt to seek medical attention until 1½ hours post-accident, that his Glasgow Coma Scale was 15, and that brain scans have been negative. Dr. Dees opined that it was not, even though these would be indicators as to extent of injury. He claimed that symptoms could emerge and/or become more pronounced and the "full effects of the injury may not be evident for days or weeks after the injury/impact." It is well understood that the brain is most dysfunctional immediately after injury, and symptoms are more pronounced shortly after injury than later. Dramatic worsening of symptoms remote from injury would not reflect a trauma-related neurologic process, unless the plaintiff had experienced subsequent brain bleeding or seizures, neither of which occurred. Worsening of symptoms over time is a red flag for psychiatric overlay and/or deliberate misrepresentation of symptoms.

4) Dr. Dees was asked whether it was appropriate for the plaintiff to have terminated his professional license and to have stopped working as a psychologist, and whether he would be able to return to his pre-injury function. Dr. Dees opined that it was unlikely that the plaintiff could ethically return to his professional duties. However, again there are several problems with this conclusion, including: 1) there are no long term cognitive sequelae from the type of injury sustained by the plaintiff, 2) it was not verified that the plaintiff was performing to true ability on the testing because no PVTs were administered, and 3) no appropriate personality testing was conducted

that would have provided information as to whether the plaintiff's symptom report was accurate or not, and to rule out symptom over-report due to a somatoform condition.

The plaintiff underwent counseling and cognitive rehabilitation with a speech pathologist and psychology intern, John Cooper, M.A., on December 21, 23, and 28, 2015; January 4, 7, 11, 14, 18, 21, 25, and 28, 2016; February 4, 8, 11, 15, 18, 22, and 25, 2016; March 3, 7, 10, 17, 21, and 28, 2016; April 4, 11, 18, and 29, 2016; May 9, 16, 23, and 30, 2016; June 21 and 27, 2016; July 25, August 1, 15, and 22, 2016; September 19 and 26, 2016; October 10, 24, and 31, 2016; November 21 and 28, 2016; December 5 and 12, 2016. On May 30, 2016, the plaintiff was describing that he was "*continuing to decline*" since the 2015 accident, and that he was feeling increasingly depressed; he became tearful describing friends who died in Viet Nam. On August 1, 2016, the plaintiff was reporting that he thought his use of opioid medications was worsening his memory and social isolation (that he had become increasingly isolated over the previous months). On August 22, 2016, the plaintiff relayed that "many of his experiences while in combat continue to haunt him. He discussed specific incidents when he feared that he had taken actions that may have brought harm to others in the wartime setting, and discussed his intense sadness over the loss of many of his friends in combat." As of October 10, 2016, the plaintiff was indicating a desire to "take a sabbatical away from his work as a diagnostic psychologist." On October 24, 2016, the plaintiff reported the death of his son, and was describing inability to cope, hopelessness, sadness, depression, and numbness; speech was observed to be normal in rate, rhythm, and articulation, and language skills were within normal limits. The plaintiff was also describing that he had been "notified of a problem with one of his recent diagnostic reports, and has chosen to suspend his psychologist license at this time."

Mr. Cooper was also asked by plaintiff counsel to answer the same questions posed to Dr. Dees, to which he provided the following responses in a letter dated February 16, 2017.

1) Mr. Cooper was asked whether the symptoms he observed in the plaintiff were consistent with the history of a closed head injury sustained on October 13, 2015. Mr. Cooper responded that they were expected for that injury based on his 15-year history of working with survivors of TBI, but in fact research shows, as summarized above, that there are no long term cognitive sequelae from the type of injury sustained by the plaintiff. Additionally, without administration of PVTs and use of appropriate personality testing to check for accuracy of symptom report and to rule out presence of a somatoform condition, it cannot be assumed that the plaintiff's claims of dysfunction are accurate.

2) Mr. Cooper was asked whether the plaintiff's history of premorbid minimal difficulty with memory, PTSD, mild depression, and some difficulty with focus contained in the records from the plaintiff's family physician could explain the plaintiff's current symptom report. Mr. Cooper opined that the plaintiff's cognitive problems were exacerbated by the 2015 accident. However, the plaintiff is engaging in hyperbole regarding his post-accident cognitive difficulties (i.e., that they are "severe" and "horrible" when in fact most scores are fully intact and the areas of lowered scores tend to be low average).

3) Mr. Cooper was asked whether it was significant that EMS at the scene of the October 2015 accident were told that there were no injury and that EMS were not needed, that the plaintiff did not attempt to seek medical attention until 1½ hours post-accident, that his Glasgow Coma Scale was 15, and that brain scans have been negative. Mr. Cooper opined that it was not, even though these would be indicators as to extent of injury. The brain is most dysfunctional immediately after injury, and symptoms are more pronounced shortly after injury than later. As summarized above, the plaintiff's injury characteristics (specifically, lack of evidence of injury) immediately after the accident, as shown by meta-analytic research, are associated with no long term cognitive sequelae. Mr. Cooper opined that brain swelling can occur hours after a head injury, but brain imaging would have detected brain swelling.

4) Mr. Cooper was asked whether it was appropriate for the plaintiff to have terminated his professional license and to have stopped working as a psychologist, and whether he would be able to return to his pre-injury function. Mr. Cooper opined that it was unlikely that the plaintiff could safely and ethically return to his professional duties. However, again there are several problems with this conclusion, including: 1) there are no long term cognitive sequelae from the type of injury sustained by the plaintiff, 2) it was not verified that the plaintiff was performing to true ability on the testing because no PVTs were administered, and 3) no appropriate personality testing was conducted that would have provided information as to whether the plaintiff's symptom report was accurate or not, and to rule out symptom over-report due to a somatoform condition.

Sandia Neuropsychology

The plaintiff underwent neuropsychological evaluation on May 30 and June 1, 2018, with Robert Thoma, Ph.D. It was concluded that the plaintiff performed "significantly below expectation in reaction time, motor speed, sustained attention, learning and memory, and confrontation naming." The plaintiff was diagnosed with "Major Neurocognitive Disorder due to Traumatic Brain Injury, mild to moderate severity, with behavioral disturbance." The plaintiff endorsed moderate symptoms of depression, and minimal symptoms of anxiety, and was also diagnosed with "unspecified Adjustment Disorder precipitated by the 2015 accident." However, there are numerous problems with these conclusions, as follows:

1) Only two performance validity tests (PVTs) were administered (one on day 1 of the testing, and one on day 2), which falls below current practice in the field. As discussed previously, the average number of PVTs utilized by practicing neuropsychologists in a forensic context is six (Martin et al., 2015) and to employ a single PVT across several hours of examination (per day) is unacceptable. Further, no actual test scores are reported for the two PVTs; therefore it is not possible to confirm that the plaintiff in fact passed the measures. Additionally, both PVTs involved assessment of validity of visual memory test performance, and would not necessarily provide data regarding veracity of test performance in other cognitive domains, such as verbal memory, processing speed, motor function, attention, and

naming. In fact, the plaintiff likely fell below cut-offs used to assess for performance validity on two tests administered by Dr. Thomas: the finger tapping test (3rd percentile score; Arnold et al., 2005) and the word list learning task (6 false positive errors on CVLT-II; see Boone et al., 2005 for recognition false positives on a similar measure, the RAVLT). Without adequate sampling of performance validity, Dr. Thoma does not have verification that the plaintiff was in fact performing to true skill level, and low scores from that exam cannot be used as evidence of cognitive dysfunction.

2) Virtually all of the reported lowered test scores have been found in research not to be lowered on a long term basis in mild traumatic brain injury/concussion. For example, lowered performance on the WAIS-IV Logical Memory subtest is not observed, even in patients with mild complicated or moderate traumatic brain injury (Carlozzi, Grech, & Tulsky, 2013); of note, Dr. Thoma reported using normative comparison data on this test for individuals age 60-69 "for direct comparison with the 2016 evaluation," but comparing a person in his 70s with a 60-69 age group will "overpathologize" the results because older individuals have worse memory skills than younger individuals; the correct normative data to have been used was that for individuals age 70-79. The plaintiff was also reported to have shown a particularly poor reaction time on the Connors' Continuous Performance Test – Second Edition (CPT-II), but patients with traumatic brain injury do not differ from normal controls on this score, including even patients with moderate to severe traumatic brain injury (Zane et al., 2016). These authors stated "we cannot conclude that our results indicate some form of permanent impairment in persons with M-TBI [mild traumatic brain injury]. In general, minimal differences in CPT-II performance were seen between the healthy control group and the M-TBI group" (p. 1002). Likewise, on the 2018 exam the plaintiff was described as performing poorly (borderline range) in rapid color naming, but lowering of this skill is not found in concussion/mild traumatic brain injury (see Dikmen et al., 1995). Similarly, the plaintiff was described as obtaining primarily low average, and even borderline, scores on a word list learning task (CVLT-II), but lowered performance on this test is not found in patients with remote concussion (Etterhoffer & Abeles, 2009). On Dr. Thoma's exam the plaintiff was now observed to have a bilateral tremor that was not reported by previous evaluators, and on 2018 testing he showed borderline impaired finger dexterity that was not documented on previous exams. Dr. Thoma suggests that the tremor is due to injury to the pontine-cerebellar motor control system in the accident, but if so, why did he not show the symptoms prior to 2018? Of note, significantly lowered finger tapping scores are an indicator of performance invalidity (see Arnold et al., 2005), particularly given that reduced finger tapping speed does not occur in the context of remote concussion (see Dikmen et al., 1995). If in fact the plaintiff has deficits in these above tests, it would not be due to an equivocal concussion in 2015. In this vein it is relevant that the plaintiff was observed on exam in 2018 to have "frequent word finding problems" that interfered with speech fluency, some slurring of speech sounds, and that he spoke so softly that at times he was not understandable." These speech characteristics were not observed by any previous evaluator closer in time to the 2015 accident. Thus, to the extent that the plaintiff actually has these speech characteristics, they would not be due to the accident in 2015. As discussed earlier,

symptoms of traumatic brain injury are most pronounced at the time of injury and then either gradually improve or remain static, and there would be no brain/behavior mechanism to account for escalating symptoms remote from injury.

3) As summarized earlier, numerous meta-analyses regarding outcome from concussion show no longterm cognitive abnormalities. As discussed above, if the plaintiff does have any actual current cognitive inefficiency, he has other conditions which would better account for cognitive abnormalities than a remote equivocal concussion, such as sleep apnea, depression, and chronic use of prescribed sleep medication (particularly in combination with reportedly drinking 1 to 1.5 glasses of wine 4 to 5 days per week). Additionally, the plaintiff reported to Dr. Thoma that he was prescribed Ritalin in college for attentional problems, and this, particularly in combination with mediocre grades in school (B and C grades in elementary school and high school, 3.2 GPA in college), raises the likelihood of a pre-existing attention deficit disorder; adults with histories of attention deficit disorder childhood continue to show cognitive abnormalities in adulthood (see meta-analysis by Schoechlin & Engel, 2005).

4) As with the 2015 and 2016 exams, no appropriate personality testing was completed. Self-reporting symptom rating scales were employed (Beck Depression Inventory – II; Beck Anxiety Inventory) that contained no validity scales to confirm that the plaintiff was reporting symptoms in a credible manner. Further, only depressive and anxiety symptoms were assessed, and no testing was employed to determine whether the plaintiff might have psychiatric conditions which could account for his reported long term concussion symptoms, such as a somatoform disorder. As discussed above, to the extent that the plaintiff actually has depression and/or anxiety, these would not be related to an equivocal concussion in 2015 per meta-analytic research. Further, Dr. Thoma did not appear to be aware that the plaintiff's son died in 2016, which would likely have a more major impact on current depression than an equivocal concussion in 2015.

5) Dr. Thoma is not board certified in neuropsychology, which raises questions as to his actual level of competency in neuropsychological assessment, i.e., no official neuropsychological organization has vouched as to his competency in the field.

Mindset

In a report issued by Drs. Jeffrey Lewine and Erin Bigler (undated, but which references exams completed in June of 2018) and incorporating the neuropsychological findings of Dr. Thoma, they opine that "whereas many of his current test scores are in the average range, these most likely indicate substantial compromise relative to premorbid abilities." However, this is not accurate. In research from my lab at Harbor-UCLA Medical Center (Palmer et al., 1998), in a sample of normal individuals with high average IQ, three-fourths obtained at least one borderline to impaired score across a neuropsychological battery, and 20% of them obtained at least two impaired scores. In a fascinating study of college professors (Zakzanis & Jeffay, 2011) with an average IQ in the superior range (FSIQ = 124) and negative medical and psychiatric histories, 30% were found to have at least one low average score, 10% achieved at

least one borderline score, and an impaired score was documented in 15%. Multiple studies have shown that individuals with high intelligence do not obtain uniformly elevated scores on cognitive exam (Diaz-Asper, Schretlen, & Pearlson, 2004; Hawkins & Tulsky, 2001; Russell, 2001), leading Greiffenstein (2008) to conclude that the belief that above average scores should be consistently found across cognitive tasks in individuals with above average IQ is a neuropsychological "myth."

Drs. Lewine and Bigler note that the plaintiff had undergone brain MRI in 2013 due to complaints of memory decline at that time, which revealed ischemic changes; research from my lab demonstrated that once white matter hyperintensities exceed a threshold amount, significant cognitive decline emerges (Boone et al., 1992). Ischemic microvascular brain changes are related to such conditions as high blood pressure, and in this vein, it is relevant that the plaintiff, in his deposition, reported that his blood pressure is controlled "sometimes." Drs. Lewine and Bigler indicate that the plaintiff's son had moved in with him post-accident to "assure that bills were paid on time and that his home environment remained clean and safe," however, by the time of the plaintiff's deposition less than two months later, the plaintiff testified that his son had moved to his own apartment (which prompted the exclamation "Thank God!").

Various electrophysiological techniques were used to evaluate the plaintiff at the Mindset facility, which were described as showing "severe" abnormalities. Brain MRI was judged to show a "dramatic change" in brain and hippocampal volume from imaging obtained nine days after the accident to June of 2018, and it was concluded that the plaintiff was experiencing "neurodegeneration" triggered by the accident in October of 2015. It was suggested that the plaintiff was at increased risk for the development of Alzheimer's disease and post traumatic epilepsy, and that there were concerns that the plaintiff "will soon be unable to safely care for himself without significant accommodations and assistance." However, imaging changes would have been most pronounced shortly after injury, and then would have remained static, or more likely, improved over time; they would not have worsened over time. "Neurodegeneration" is not triggered by an equivocal concussion. Further, research shows no increased risk of Alzheimer's disease or seizures after concussion (Singer, 2001; Plassman et al., 2000; Godbolt et al., 2014; Crane et al., 2016).

When evaluating "damages" in a lawsuit, what has to be demonstrated is loss of functionality, which is why the results of neuropsychological and psychological testing are paramount because they address decrements in function. Dr. Bigler has in fact previously argued that "neuropsychological testing defines the neurobehavioral significance of neuroimaging-identified abnormalities," and in his 2001 publication he described a case of a teacher who sustained a severe traumatic brain injury, with well-defined structural abnormalities on imaging ("prominent left frontal lesion"), and who subsequently completed a masters degree. The cases contained in Dr. Bigler's publication well illustrate that there is not a one-to-one correlation between imaging findings and neuropsychological test results and "real world" functionality. In fact, research has shown that 18% to 21% of asymptomatic individuals who undergo head MRI for reasons unrelated to brain function are found to have intracranial abnormalities (Katzman et al., 1999; Kim et al., 2002), and such incidental findings are observed to increase with age (Morris et al., 2009). Further, research shows that the various conditions diagnosed in the plaintiff, such as sleep apnea (Canessa et al., 2011; Yaouhi et al.,

2009), depression (Anand et al., 2005; Lorenzetti et al., 2009), and hypertension (Beason-Held et al., 2007; Salerno et al., 1992), are associated with functional and structural brain changes, and these would better account for any abnormalities identified at the Mindset facility.

Seton Family of Hospitals

The plaintiff presented at Seton Southwest Hospital on October 13, 2015, at 4:09pm via private vehicle. He was reporting left neck and left-sided body pain. He was observed to be alert and oriented. His reported medications included zolpidem and alprazolam. Head CT was normal.

University Medical Center Brackenridge

The plaintiff presented at the University Medical Center Brackenridge on October 13, 2015, at 9:12pm. He was ambulatory and unaccompanied. He was describing neck and back pain and numbness in the 4th and 5th fingers of his left hand secondary to a head on collision. He reported that his current medications were zolpidem, alprazolam, terazosin, and lisinopril. He was observed to be alert and oriented with steady gait; Glasgow Coma Scale was 15, and speech was clear. He left at 11:16pm without being evaluated.

ARA Diagnostic Imaging

Brain MRI obtained on October 22, 2015, was normal with the exception of a few scattered foci of white mater signal abnormality related to minor small vessel ischemic damage.

Neuroaustin/Andrea Raymond, M.D.

In the initial evaluation on October 15, 2015, the plaintiff reported that he had been involved in a motor vehicle collision two days earlier. He indicated that his airbag deployed and he did not lose consciousness. He described subsequent persistent sleep disturbance, memory/cognitive issues, “weepiness” at the scene, dizziness, headaches, feelings of stress, and difficulty concentrating, but no anxiety, depression, or personality change. He worked the day following the accident, and reported that he was slower than normal and “had to take a lot of notes but was able to get through the day.” He indicated a longstanding history of back pain for which he had been prescribed norco for six years but not in the last 1.5 years; he had again been prescribed norco for neck pain on October 14, 2015. He described a past history of migraines, chronic insomnia, hypertension, and lower back and neck pain, and that he had seen a mental health provider in 1984. His medications as of October 15, 2015, were lisinopril, alprazolam, and trazadone. On follow up on October 30, 2015, the plaintiff was reporting postconcussive symptoms including memory loss, cognitive slowing, excess sleeping, anxiety, difficulty concentrating, and mild depression. He relayed that he was performing worse on Luminosity, particularly those tasks involving short term memory. He obtained a score of 23/30 on the MOCA; he recalled 0 of 5 words on delayed recall, only generated 6 words beginning with the letter B in 1 minute, and did not know the day of the month. His medications included alprazolam and trazodone. On follow up on December 10, 2015, the plaintiff was reporting postconcussive syndrome, feelings of stress, difficulty concentrating, and sleep disturbance, but was denying depression, anxiety, and personality change. His medications included duloxetine, Remeron, alprazolam, and trazodone. On follow up on January 14, 2016, the

plaintiff was continuing to describe postconcussive symptoms, anxiety, feelings of stress, personality change, difficulty concentrating, and sleep problems; he was denying depression. He had been prescribed duloxetine and "did not like how he felt on it" and discontinued it. Additional medications included norco, Remeron, and alprazolam. He was working, but stated that reports that used to take three hours now took one week. He was attending cognitive therapy. On follow up on March 1, 2016, the plaintiff was reporting neck pain, postconcussive syndrome, feelings of stress, difficulty concentrating, and sleep disturbance; he was denying depression, anxiety, and personality change. It was noted that he had been prescribed Cymbalta, "but could not tolerate it" and it was discontinued. He was participating in cognitive therapy two times per week, and taking fluoxetine three to four times per week. Additional medications included zolpidem, norco, and alprazolam. He was continuing to practice as a psychologist on a limited basis. Medications as of March 7, 2016, included fluoxetine, zolpidem, norco, and alprazolam. On April 12, 2016, the plaintiff was reporting postconcussion symptoms, as well as difficulty concentrating and sleep disturbance, but not anxiety, depression, feelings of stress, personality change. His MOCA score was 27/30 (recalled 2 of 5 words). He was prescribed lamotrigine, fluoxetine, norco, and alprazolam among other medications. On follow up on June 6, 2016, the plaintiff presented with continuing postconcussive symptoms, feelings of stress, personality change, difficulty concentrating, disorganization, and sleep disturbance; he was denying anxiety and depression. He was working, but in a limited capacity. He had been instructed to take lamotrigine due to an abnormal EEG, but had discontinued the medication because of "not liking how I felt on it." Additional medications included zolpidem, norco, alprazolam, and Ritalin. The plaintiff underwent evaluation on June 29, 2016, in the context of reported left leg weakness and chronic low back pain. Testing revealed femoral mononeuropathy and possible chronic L5 radiculopathy on the left side. On follow up on September 29, 2016, it was reported that the plaintiff had undergone back surgery in August. He was reporting postconcussive symptoms, feelings of stress, personality change, difficulty concentrating, and sleep disturbance; he was prescribed norco and Ritalin among other medications. EEG on October 3, 2016, was judged to be abnormal, with left temporal slowing and rare sharps. On follow up on October 27, 2016, he reported continuing memory loss, depression, feelings of stress, difficulty concentrating, and sleep disturbance. On follow up on December 1, 2016, the plaintiff continued to describe memory loss. It was noted that he was grieving the death of a son in October (although this was not referenced in the October record), for which he was seeing a psychotherapist. He had reportedly been placed on Ritalin in the spring of 2016. He indicated that he had been taking prescribed lamotrigine, but could not provide the dose or schedule.

The concern with these records is that as of October 30, 2015, the plaintiff arguably scored comparably to a dementia patient (Alzheimer's disease) in his word recall (0 of 5 words on delay) and in rapid word generation (6 words in 1 minute). This is inconsistent with symptoms after concussion, and inconsistent with the fact that the plaintiff was living independently, including managing his own medications, and continuing to practice as a psychologist. Over the various follow up visits the plaintiff was reported to be on a variety of medications, which certainly in combination could be expected to impact cognition, including norco, zolpidem, trazodone, and Xanax, and Ritalin could be expected to interfere with sleep. Psychiatric symptoms waxed and waned, and the plaintiff appeared to have significant stressors including episodes of leg weakness leading to falls and which resulted in back surgery in August of 2016, and the death of a son in October of 2016 for which he was described as grieving as of

December of 2016. He claimed sleep disturbance secondary to the October 13, 2015 accident, but had a history of chronic insomnia for which he was prescribed Xanax and trazodone at the time of the accident. He was also claiming headaches from the accident, but had a premorbid history of migraines. The plaintiff's report of unpleasant side effects to multiple medications would be consistent with the presence of a somatoform disorder.

Texas Orthopedics Sports and Rehabilitation Associates

The plaintiff was seen on November 19, 2015, for low back pain; no other symptoms were reported and the plaintiff was denying headaches and dizziness or any other neurologic symptoms. Medical history was noteworthy for hypertension and PTSD. Medications included alprazolam and norco. The plaintiff was again seen on December 13, 2015, at which time he was reporting cognitive problems, including memory loss, in the context of post-concussion, as well as depression. The plaintiff was seen again on March 8 and 23, 2016, with continuing report of pain, but he was denying headaches and dizziness; cognitive complaints continued to be reported. On April 15, 2016, the plaintiff presented for follow up related to epidural treatment in relation to MRI findings of L4-5 disc protrusion with displacement of the left L5 nerve root and compression of the left L4 nerve root, as well as narrowing at the L3-4 foramen; he was reporting that a few days previously his left leg had given out on him. He was denying depression, anxiety, or difficulty sleeping; mental status was normal. He again returned on July 6, 2016 with worsening of back pain, and a fall related to his left leg "giving out." It was noted that he was "very concerned about his ongoing symptoms and always being afraid that he will fall again." He continued to be prescribed norco and alprazolam, and continued to deny depression, anxiety, or sleep problems; mental status was normal.

Texas Orthopedics Surgery Center

The plaintiff underwent epidural steroid injections on November 19 and December 30, 2015, and March 10 and 24, 2016.

Neurotexas

On evaluation on July 26, 2016, the plaintiff was described as having eight years of intermittent low back pain exacerbated in a motor vehicle accident in October of 2015, as well as three episodes in which he was unable to move his left leg and fell. He was noted to be a daily smoker. Neurologic exam was normal. The plaintiff underwent L2-L5 surgery on August 5, 2016. On follow up on September 28, 2016, he was reporting that back and leg pain were improved, and he was observed to exhibit normal memory, language, and fund of knowledge. He was prescribed various medications including fluoxetine, lamotrigine, acetaminophen-codeine, zolpidem, alprazolam, and hydrocodone-acetaminophen.

Lakeway Regional Medical Center

The plaintiff underwent back surgery for degenerative scoliosis and lumbar stenosis at Lakeway Regional Medical Center on August 5, 2016, and was discharged into his own care on August 8, 2016. He exhibited some odd behavior post surgery, including claiming that someone had stolen his shoes, that he wanted his foley catheter removed and that he would

call an ambulance if he needed to void, and that he "rambles about something that is totally inappropriate." Hospital records refer to additional diagnoses of chronic PTSD, chronic insomnia, postconcussion syndrome, hypertension since 1995, chronic low back pain since 2005, hyperlipidemia since 2009, shrapnel fragments wounds in 1969, and coronary artery disease and heart catheterization in December of 2013. It was noted that the plaintiff's mother had died at age 62 of a stroke, that his father died at age 75 of an MI, that his brother had hypertension, and that his sister died of complications from pneumonia. The plaintiff was described as working part-time. He reported drinking four times per week. He additionally reported reduced libido and ED, minimal difficulty with memory and some difficulty focusing. No obvious neurologic abnormalities were detected on exam, and the plaintiff was judged to have normal memory, language, and fund of knowledge.

Encompass Home Health

Records from August of 2016 for care provided after his back surgery indicate that the plaintiff was living alone and able to manage his own medications. He was screened for depression and found to have no depressive characteristics. He was described as alert/oriented, able to focus and shift attention, and to comprehend and recall task directions independently. He was found to have no abnormal neurological findings.

Texas Heart and Vascular

Records from July 28, 2016, indicate that plaintiff had experienced episodes of high blood pressure, as well as some anginal discomfort and EKG changes. Cholesterol was still elevated; it was discussed that Crestor had seemed to cause memory difficulties. The plaintiff was diagnosed with coronary artery disease, abnormal ECG/EKG, palpitations, fatigue, dyspnea, chest discomfort, hypertension, and hyperlipidemia. The plaintiff was also seen on February 24, 2014, at which time he was reporting angina with EKG changes. When seen on January 23, 2013, the plaintiff reported that he had developed "significant memory problems" with use of Crestor.

Charles Mallett, M.D.

The plaintiff was treated by Dr. Mallett from 2012 up through at least December of 2016. All of the records from January of 2012 up through December of 2016 refer to "minimal difficulty with memory…[and] some difficulty focusing." The plaintiff discontinued crestor in March of 2012 because he thought it was causing memory loss. He was identified as having low testosterone in October of 2012, as well as ED, and had at least one testosterone injection. As of February of 2012 he was reporting that he only slept four hours daily, and in February of 2013 it was recorded that a CPAP machine had been recommended for sleep apnea. The plaintiff reported that he had not slept well since his 20s, and that his brother had sleep apnea. In June of 2013 the plaintiff was describing loss of ability to recall names of people he knew well, as well as headaches; brain MRI revealed scattered white matter hyperintensities judged to be most likely related to microvascular changes. In July of 2013 he was identified as having vitamin B12 deficiency which was considered possibly contributing to his memory loss; he continued to report headaches. In September of 2014 he was describing neck pain, and fluid on his left knee; he "googled" his symptoms and wondered if he had lupus. He had smoked

until 2004. He was reporting low back pain in August of 2015 for which he was taking anti-inflammatories. He was hospitalized for acute pancreatitis in April of 2016. In November of 2016 the plaintiff was described as under a lot of stress with the loss of his son in a motor vehicle accident six weeks previously; the plaintiff indicated that the October 13, 2015, accident had made his memory worse, but still was only reporting minimal difficulty with memory.

These records document concern with memory loss well before the 2015 accident, as well as insomnia, headaches, and neck and back pain. Both lowered testosterone and vitamin B12 deficiency have been shown in the research literature to be associated with lowering of cognitive ability (Beauchet, 2006; Eastley, Wilcock, & Bucks, 2000).

Tax Returns for Jerome Schmidt

The plaintiff's tax return for 2014 showed an adjusted gross income of $31,557, and gross receipts were $40,377. In 2013, his business income was $31,166, and in 2012 his gross receipts were $39,780.

Deposition of Jerome Schmidt (7/24/18)

In his testimony, the plaintiff described his education, military service, family, hobbies, exercise regimen, daily activities, and occupational background. In his work as a psychologist, he had reviewed workers compensation requests (to determine if they were reasonable and necessary); the workers had sustained injuries related to motor vehicle accidents as well as industrial accidents and exposure to chemicals. The plaintiff reported that a son died in 2017 (other records indicate this occurred in October of 2016) in a motor vehicle accident. He indicated that he had a premorbid history of back pain but was "improved" in the year prior to the 2015 accident. He stated that currently he is no longer taking prescribed pain and sleep medication. He described a longstanding history of high blood pressure which is controlled "sometimes." He indicated that he was living alone (his son had gotten his own apartment – "thank god").

Regarding the October 13, 2015, accident, the plaintiff exhibited no retrograde amnesia (failure to recall events immediately prior to the accident) or anterograde amnesia (failure to recall events immediately following the accident); he has very detailed recall of events immediately post accident, i.e., that he recalls an acrid smell from the collision, that he quickly exited the vehicle, that a Marine came over to check that the plaintiff was "OK, that he was asked by several people if he was hurt, etc.). He stated that he does not believe that he lost consciousness in the accident. He indicated that first responders recommended that he be transported by ambulance to a hospital, but he responded, "I don't know," and instead he rode in the tow truck which removed his vehicle from the scene, and then returned home in a rental vehicle. He then went to a facility (Seton Southwest) and underwent imaging but was informed that he needed an MRI scan, which they could not provide. He again returned home, and later that evening went to Brackenridge Hospital where he reported that he waited for two to three hours without being seen; he indicated that he was informed by a nurse that they were very busy and probably would not be able to see him, so he again returned home. He stated that

he did not know if he returned to work the following day (Dr. Raymond's records indicate that he did), or when he first received medical care.
Near the conclusion of the deposition (page 92) the plaintiff claimed not to recall opposing counsel asking him questions earlier in the deposition regarding medical providers he saw after the accident, and on page 98 he indicated that when he sat down for the deposition "I couldn't remember why we were here," and that he did not remember that they were to discuss the motor vehicle accident. On page 101 he testified that he had watched local news that morning and "I couldn't tell you one thing on it that I looked at this morning." If accurate, these severe memory lapses, such as observed in Alzheimer's disease, would have precluded him from being able to give deposition testimony (i.e., if he cannot recall why he is there). Despite these claims of not knowing why he was at the deposition, the content of questions asked earlier in the depo, and what he had seen on TV that morning, he had driven to the deposition (which requires grossly intact memory), and he recalled that it was 90 degrees (page 109), and that he had nearly rear-ended someone the previous day which caused him to "feel scared last evening" (page 108). How does he recall this information but not why he is at the deposition? When asked if his short term memory has changed since the accident (gotten better or worse) he said that he didn't know (page 101), but earlier (page 98) he had described "big holes in my memory as I go along that I just can't remember what's going on."

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In summary and conclusion,

1) There is no evidence that the plaintiff sustained a brain injury in the 2015 accident, and even if he had sustained a concussion, he would have no cognitive or psychiatric residuals from that event.

2) None of the neuropsychological exams of the plaintiff adequately checked for performance validity (i.e., confirmed that the plaintiff was in fact performing to true ability). Without documentation of valid test performance, through the plaintiff being administered, and passing, numerous performance validity tests across the several hour examinations, low scores on standard neuropsychological measures cannot be used as evidence of cognitive dysfunction.

3) If the plaintiff does have any actual mild cognitive inefficiency, he has other conditions that would more likely account for these symptoms, including sleep apnea, depression, hypertension, elevated cholesterol, lowered testosterone, vitamin B12 deficiency, small vessel vascular disease, chronic prescribed sleep medication use (particularly in combination with concurrent use of alcohol), and possible premorbid attention deficit disorder. Worsening of cognitive and physical symptoms are not seen after concussion and, if accurate, is either due to the above conditions, or raises the possibility of a separate neurologic condition.

4) Appropriate personality testing was not conducted that would have accurately assessed levels of depression, anxiety, psychological distress, PTSD, and other conditions, such as somatoform disorder. The plaintiff's over-statement of, and over-reaction to, symptoms would be consistent with a somatoform disorder, in which the individual channels stress into physical symptoms, magnifies the

seriousness of minor symptoms, and presents him- or herself as more dysfunctional than is actually the case.

The expert opinions contained in this report are provided with a reasonable degree of neuropsychological certainty, and are based on my education and experience in clinical neuropsychology, review of plaintiff's records, and authoritative research. My opinions may be supplemented or amended if additional relevant information becomes available through discovery or other experts.

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